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Case Report

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# Death from caustic ingestion: A case report

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## ABSTRACT

**Rationale:** Acute caustic ingestion from suicidal intent is not usual in emergency departments in developed countries. One of the substances commonly ingested by suicidal patients, phosphoric acid, tends to cause multi-system derangements.

**Patient's Concern:** A 41-year-old male patient presented with complaints of throat discomfort, severe generalized abdominal pain, and multiple episodes of hematemesis after ingesting a restroom cleaning solution.

**Diagnosis:** Poisoning by acute caustic ingestion (containing <30% phosphoric acid and <4% ethylene glycol).

**Interventions:** The patient was administered 50 mL of 8.4% sodium bicarbonate solution followed by an isotonic sodium bicarbonate solution running at 500 mL/h, a hyperkalemia kit, ceftriaxone, metronidazole, omeprazole, and atropine. The patient then underwent urgent hemodialysis.

**Outcomes:** The patient suffered gastrointestinal bleeding as a result of local caustic injury. In addition, his course of illness was complicated by severe acidemia from high anion gap metabolic acidosis and deranged electrolytes (hyperphosphatemia, hyperkalemia, and hypocalcemia). He developed multi-organ failure and eventually demised.

**Lessons:** The clinician needs to be mindful of the multi-system complications arising from such a caustic ingestion. These patients need to be monitored closely for deterioration, and have prompt management of the various arising complications, to reduce the high morbidity and mortality associated with this condition.

**KEYWORDS:** Phosphoric acid poisoning; Caustic ingestion; Acid ingestion; Cleaner ingestion; Corrosive ingestion

## 1. Introduction

Acute caustic ingestion from suicidal intent is not usual in emergency departments in developed countries. Patients with this condition commonly present with severe abdominal pain and multi-system derangements, such as deranged electrolytes and acid-base imbalances, potentially leading to multi-organ failure and resulting in high morbidity and mortality. This study reports a case of a patient with such an ingestion and management of the complications that arose from it.

## 2. Case report

This case report was approved by our hospital's local ethics committee and written in accordance with our institutional guidelines. Consent was taken from the appropriate involved parties and the authors do not report any conflict of interest in preparation of this manuscript.

A 41-year-old male patient presented to the emergency department three hours after drinking 1 cup of Maintex Lime Go restroom cleaner (containing <30% phosphoric acid and <4% ethylene glycol) in a suicidal attempt. He presented with complaints of throat discomfort, severe generalized abdominal pain, and multiple episodes of hematemesis. He had no significant past medical history.

On arrival, he was afebrile, with a heart rate of 79 beats/min (normal: 60-100 beats/min), a blood pressure of 131/89 mmHg (normal: 120/80 mmHg), and a respiratory rate of 19/min (normal:

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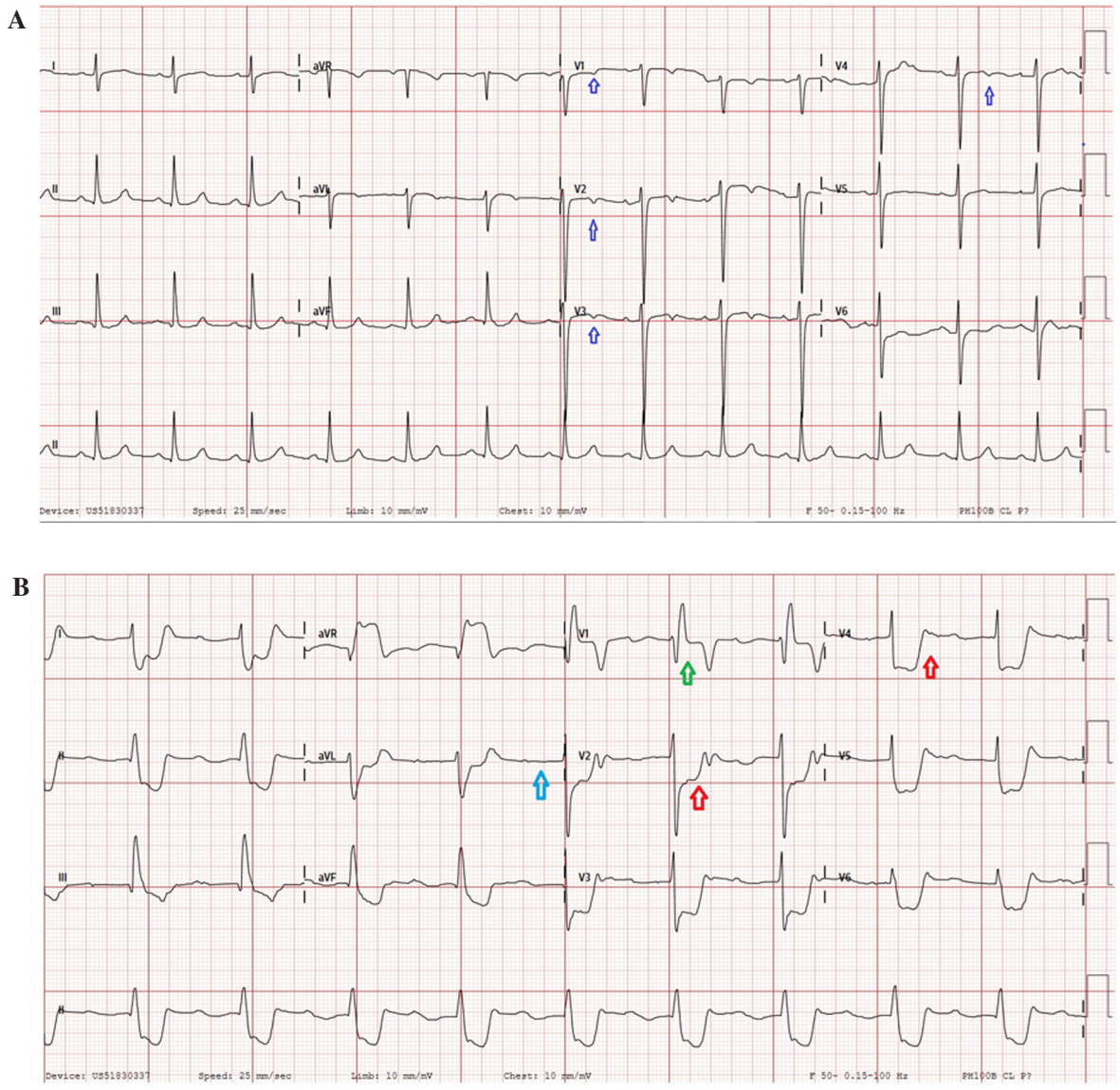


**Figure 1.** The 12-lead electrocardiogram of a 41-year-old patient showing sinus rhythm with T inversions over the anteroseptal leads (blue arrows) (A). The patient's repeat electrocardiograms revealing a sinus bradycardia with prolonged PR interval (blue arrow), right bundle branch block (green arrow) and left posterior fascicular block, and diffuse ST depressions (red arrows) (B).

12-18/min). On physical examination, he was found to be alert but in pain. His throat was mildly erythematous and his heart and lung examinations were normal. He had generalized abdominal tenderness without rebound or guarding. Bowel sounds were present. Altered blood was noted in his stools.

Laboratory investigations revealed the following: white blood cell count of $31.2\times10^9$/L (normal: $4\times10^9$/L-$10\times10^9$/L), hemoglobin of 16.7 g/dL (normal:12-16 g/dL), and platelet count of $206\times10^9$/L (normal: $140\times10^9$/L-$440\times10^9$/L). His serum urea was 3.5 mmol/L (normal: 2.5-7.8 mmol/L) and creatinine was 102 μmol/L (normal: 45-84 μmol/L). Serum electrolytes were as follows: sodium 139 mmol/L (normal: 133-146 mmol/L), potassium 5.4 mmol/L (normal: 3.5-5.1 mmol/L), chloride 95 mmol/L (normal: 95-110 mmol/L), magnesium 0.98 mmol/L (normal: 0.7-1.0 mmol/L), corrected calcium 1.98 mmol/L (normal: 2.1-2.6 mmol/L) and phosphate 12.92 mmol/L (normal: 0.94-1.5 mmol/L). His serum pH was 7.007 (normal: 7.35-7.45), serum bicarbonate was 5.7 mmol/L (normal: 22-29 mmol/L), base excess was −21 mmol/L (normal: −2-3 mmol/L), serum glucose was 11.2 mmol/L (normal: 3.9-11 mmol/L) and serum lactate was 3.3 mmol/L (normal: 0.5-2.2 mmol/L). His anion gap was 38.3 mmol/L (normal: 4-12 mmol/L). His coagulation profile was as follows: activated partial thromboplastin time was 45.2 s (normal: 25.7-32.9 s), prothrombin time was 13 s (normal: 9.9-11.4 s) and international normalized ratio was 1.26 (normal: <1.1). His liver function test was unremarkable apart from a slightly elevated alkaline phosphatase of 118 U/L (normal: 35-104 U/L). His

serum lipase was 155 U/L (normal: 13-60 U/L). Paracetamol and salicylate levels were undetectable.

His initial 12-lead electrocardiogram showed sinus rhythm with T inversions over the anteroseptal leads (Figure 1A). He subsequently developed bradycardia with a heart rate ranging between 45-55 beats/min and repeat electrocardiograms revealed sinus bradycardia with prolonged PR interval, right bundle branch block, left posterior fascicular block, and diffuse ST depressions (Figure 1B). A chest radiograph did not show any focal consolidation, pneumothorax, pleural effusion, pneumo-mediastinum, or subphrenic free air. A computed tomography chest, abdomen, and pelvis revealed fluid-filled and borderline distended small and large bowel loops, likely related to the recent caustic injection. There was no obstruction, overt bowel wall thickening, or perforation. Naso-endoscopy performed by the otolaryngologist team at the bedside did not reveal any signs of upper airway injury, such as edema or erythema. The initial issues identified were caustic ingestion, leading to caustic oesophageal and gastric injury with gastrointestinal bleeding, complicated by severe high anion gap metabolic acidosis with hyperkalemia, hyperphosphatemia, and hypocalcemia.

He was placed on 3 L nasal prongs and cardiac monitoring. He was treated with 50 mL of 8.4% sodium bicarbonate solution followed by an isotonic sodium bicarbonate solution running at 500 mL/h. A hyperkalemia kit consisting of 10 units of rapid-acting insulin (Actrapid), 40 mL of 50% dextrose, and 2 cycles of 10 mL of 10% calcium gluconate was also given. He was given a total of 10 mg of morphine for pain control and covered with empirical ceftriaxone and metronidazole. Symptomatic medications including metoclopramide and omeprazole were also given. A total of 2.4 mg of atropine was given in boluses of 0.6 mg to correct bradycardia.

After receiving the treatments mentioned above in the emergency department, he was then admitted to the intensive care unit (ICU) for urgent hemodialysis. During the ICU stay, the patient deteriorated further, with peri-arrest from severe electrolyte derangement and bradycardia. He developed mixed cardiogenic and hypovolemic shock requiring high-dose vasopressors. He also developed acute liver failure from ischaemic hepatitis and phosphate toxicity, complicated by disseminated intravascular coagulation.

Despite undergoing dialysis, his condition worsened, with repeated episodes of gastrointestinal bleeding requiring multiple blood transfusions and high-dose proton pump inhibitors. Endoscopy revealed severe oesophagitis, pangastritis, and duodenitis. A computed tomography aortogram did not reveal any aorto-enteric fistula. His condition deteriorated substantially with time and eventually led to multi-organ failure and his demise.

## 3. Discussion

Acute caustic ingestion is not uncommonly seen in emergency departments in developed countries, with ingestion of household cleaning substances being responsible for slightly above 7% of all poison exposure cases based on the 2022 annual report of the American Association of Poison Control Centers' National Poison Data System[1].

Phosphoric acid is a component of fertilizers, detergents, and many household cleaning products. Dilute solutions are also used as a food additive in soft drinks. Phosphorus is an essential nutrient that plays an important role in cellular function as a component of nucleotides, nucleic acids, and phospholipids. It is also a cofactor in many enzyme systems ranging from energy metabolism to cell signaling. During acidosis, phosphorus shifts from the intracellular to the extracellular fluid space, leading to a further rise in serum phosphate levels. A significant portion of this becomes non-filterable and forms colloidal complexes with calcium, leading to hypocalcemia[2-4].

Acute phosphoric acid ingestion may lead to acute local complications of caustic damage to the gastrointestinal tract such as esophagus-gastritis, and also cause gastrointestinal hemorrhage and gastric perforation, and delayed local complications of fistula and stricture formation[5-7]. Direct contact of the phosphoric acid with the respiratory mucosa may also lead to airway edema and respiratory failure[5]. In addition to local complications, systemic effects of severe metabolic acidosis and electrolyte derangements including hyperkalemia, hyperphosphatemia, and hypocalcemia have also been documented[3,8].

The overall management of a patient with acute phosphoric acid ingestion involves a multi-disciplinary approach. The airway needs to be evaluated early on, and secured in the event of significant injuries. As with any caustic ingestion, early endoscopy and imaging are also needed to evaluate the caustic effects on the gastrointestinal tract[9]. Toxicology consultation and aggressive medical therapy involving the use of intravenous crystalloids, intravenous sodium bicarbonate, and correction of electrolyte abnormalities are often required to address the systemic effects of metabolic acidosis and electrolyte derangements[8]. In cases of severe acute toxic ingestion, arrangement for hemodialysis in the for removing excess serum phosphate may be life-saving as well, though there is a paucity of data in the literature regarding this specific patient group[10].

## Conflict of interest statement

The authors report no conflict of interest.

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## Data availability statement

The data supporting the findings of this study are available from the corresponding authors upon request.

## Authors' contributions

SHK was involved in conceptualizing and revision of the manuscript. JCLT was involved in drafting of the manuscript.

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